MAR 1952 01-46

CLASSIFICATION S-E-C-R-E-T
SECURITY INFORMATION
CENTRAL INTELLIGENCE AGENCY
INFORMATION FROM
FOREIGN DOCUMENTS OR RADIO BROADCASTS

REPORT
CD NO.

50X1-HUM

COUNTRY GDR

SUBJECT Scientific - Medicine, veterinary, hoof-and-mouth disease

HOW PUBLISHED Semimonthly periodical

WHERE PUBLISHED Leipzig

DATE PUBLISHED 15 Apr 1953

LANGUAGE German

DATE OF INFORMATION 1953

DATE DIST. 13 Jul 1953

NO. OF PAGES 3

SUPPLEMENT TO REPORT NO.

THIS IS UNEVALUATED INFORMATION

SOURCE Monatshefte fuer Veterinaer Medzin, Vol VIII, No 8, Leipzig, 1953, pp 153-155.

TYPE TRANSFORMATION OF THE VIRUS OF HOOF-AND-MOUTH DISEASE

Prof H. Roehrer
Island of Riems

At the time when the writer was working at the Station for the Experimental Study of Hoof-and-Mouth Disease on the Island of Riems, near Greifswald, Germany, an epidemic of the disease, starting in Western Germany, was spreading over Europe, making imperative a close study of a possible transformation of types of the causative agent of the disease.

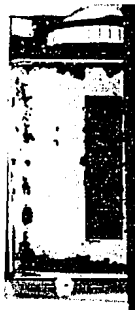
The previous great epidemic of 1937 - 1938 had affected the same region, but between 1938 and 1951, there had been only sporadic, light outbreaks, which were important because not only types A and O were found, but also type C. Vaccination soon brought the various foci under control, but since only a small percentage of animals had been exposed, those not exposed easily fell victims when the new epidemic started early in 1951. The only cattle not touched were those of the German Democratic Republic which had received prophylactic injections of A and O. (It should be mentioned here that the 1951 type A_5 is identical with type B_4, which was isolated at Riems from West German material.)

It is not known why type A_5 should suddenly show such increased virulence. It is certain, however, that the sensitivity to infection of the European cattle and the rapid passage of the virus through sensitive animals, especially through swine, greatly contributed to the increase in virulence.

There were no changes in the virus found in vaccinated and nonvaccinated calves and goats, and no variation or shift of type. During passage of the highly virulent virus through vaccinated animals, it lost much of its virulence, both in regard to contagiousness and to pathogenity. The latter, however, was retained in regions where the stock had not been protected.

CLASSIFICATION S-E-C-R-E-T

STATE	X	NAVY	X	NSRB		DISTRIBUTION											
ARMY	X	AIR	X	FBI													



In 1951, relatively soon after the end of the A_5 epidemic in West Germany, there occurred a new outbreak of the C-type disease, which soon made its appearance in the German Democratic Republic, where it had been imported from the West, as we were able to prove. At this time, type C was highly pathogenic but showed only little tendency to spread, and the foci were soon brought under control. Thus, type C showed the characteristics which had been described earlier by B. I. Kindyakov, A. N. Bayadinov, S. M. Filippovich, and O. S. Nikonova (Veterinariya, Vol XXIX, No 8, 1952). In spring 1952, another epidemic developed in West Germany, spreading over the greater part of Europe and the parts of the German Democratic Republic which were not protected against type C. This time, the virus developed everywhere a hitherto not encountered virulence.

This was not surprising since Kindyakov, Bayadinov, Filippovich, and Nikonova had described in detail how such a transformation into type C could take place if animals immune to type A and non-immune /completely receptive/ animals recently infected with type A were in close contact in the same pastures. Under the circumstances, the originally resistant immunized animials also caught the disease. It was established that type C virus was now present in the newly infected cattle. Conditions similar to those described by the Soviet authors must have existed in Western Germany.

We believe that there was no shift in type from A_5 to C in the German Democratic Republic because cattle here had been vaccinated and carefully quarantined.

We know that when an epidemic is nearing its end, that is to say, as soon as a high percentage of the stock has been infected, a change begins in the antigen structure of the virus, which leads to a break in immunity. (Magnussen and Hermansson: Report on the Swedish epidemic of 1926 - 1927, where 40% of the originally infected cattle became reinfected within 10-12 months. The Danish epidemic of 1924 - 1926 showed a similar picture; see Waldmann and Nagel, Handbuch der Viruskrankheiten, Vol I, Verl. Gustav Fischer, Jena, 1939.) All this tends to explain the appearance of type C during the 1952 epidemic.

A transformation in type should also be possible in the laboratory. The above-named Soviet authors state that by passing the virus through immune guinea pigs, a change of the C into the O type took place, and on passage of the virus through immune cattle, a change from the A into the C type was achieved. Demnitz and Geiger report that they finally obtained a transformation into the C type by injecting O virus into cattle immune against O. Up to now, such experiments have not been undertaken at Riems, because it was believed that despite the greatest precautions, contamination with some other type might take place and spoil the experiments. There has never been any indication of a change in type during the experiments, although there were repeated disruptions of these experiments due to involuntary infection with other types. As soon as our new experimental station at Greifswalder Oie, 15 kilometers from the mainland, is in operation, we shall be able to start experiments in this direction.

Nothing definite is known at present regarding the biological processes of type transformation of the hoof-and-mouth disease virus. In the last analysis, the process is biochemical and cannot be studied until research into the chemical nature of the causative agent and its propagation mechanism in correlation to the host organism is completed. At present, only conjectures are possible.

Some authors are of the opinion that with respect to the immune host animal, the virus changes its type to "safeguard its kind," but that would mean that it has capabilities which it evidently cannot have, and should be rejected as unscientific.

Kindyakov, Bayadinov, Filippovich, and Nikonova assume that after repeated passage of the virus through immune animals, there is a change of metabolic conditions, which, in the last analysis, is the cause of the virus transformation. They believe that the structure of the virus is not a homogeneous, antigenous whole but a complex of specific protein antigen fractions in various combinations and that these combinations change, depending on the conditions of the surrounding medium, the original virus type remaining in-active without being destroyed.

Nothing can be said against this assumption at this time. We may be dealing with a complex of special protein antigens, one of which, depending on the immuno-biological status of the host organism, becomes dominant. On the other hand, it is possible that chemical reactions of anti-bodies in the host organism may be the cause of the change in the molecular structure of the virus.

These fundamental questions will be answered only after more advanced study of the biochemical character of the causative agent, especially by the use of isotopes.

As far as the conclusions contained in the work by Kindyakov, Bayadinov, Filippovich, and Nikonova are concerned, the author feels that, based on present experiences in the German Democratic Republic, a type transformation need not be expected when the causative agent meets a completely vaccinated and quarantined cattle population, an opinion which is borne out by the observations reported by Kindyakov, Bayadinov, Filippovich, and Nikonova.

- E N D -